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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/695,089	CHEUNG ET AL.	
	Examiner	Art Unit	
	Stephen Kapushoc	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *27 February 2007*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-5,12 and 13* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-5, 12 and 13* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Claims 6-11 are been cancelled.
Claims 1-5, 12 and 13 are pending and examined on the merits.

This Office Action is in reply to Applicants' correspondence of 02/27/2007. Claim(s) 6-11 is/are cancelled; no claims are withdrawn; claim(s) 12 and 13 has/have been newly added; no claims have been amended.

Applicants' remarks have been fully and carefully considered but are not found to be sufficient to put the application in condition for allowance. Any new grounds of rejection presented in this Office Action are necessitated by Applicants' amendments.

This Action is made **FINAL**.

New Rejection
Claim Rejections - 35 USC § 112 1st¶ - New Matter

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 12 and 13 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This is a **NEW MATTER** rejection.

Claims 12 and 13 are drawn to methods requiring the use of primers which are broadly claimed as 'having **a** sequence as set forth in nucleotides 1 to 20 of SEQ ID NO:' 24 or 25 (for the analysis of PM1, claim12) or 66, 67, or 68 (for the analysis of PM2, claim 13). Applicants point to support for the subject matter of claims 12 and 13 in paragraphs [030], [032], [035], and [040] and in Figure 1E. However the cited portions of the specification do not provide a basis for the broadly claimed subject matter. For

example paragraphs [030] and [032] oligonucleotides suitable for the analysis of the PM1 and PM2 mutations are 'for example, an oligonucleotide comprising any one of SEQ ID NO:' 24, 25, 66, 67, or 68 (as recited in paragraphs [030] and [032]). Figure 1E indicates that oligonucleotides suitable for detection of PM1 and PM2 include oligonucleotides consisting of SEQ ID NO: 24 or 25 (PM1 reverse) and 66, 67, or 68.

Thus the specification does not provide support for the broadly claimed primers, where the use of the indefinite article 'a' in the phrase 'having **a** sequence as set forth in nucleotides 1 to 20 of SEQ ID NO:' is inclusive of any primers having as few as two contiguous nucleotides (where as few as two nucleotides is 'a sequence') of the recited SEQ ID NOs. This new matter rejection may be overcome by amendment of the claims to recite 'comprising SEQ ID NO:' instead of the phrase 'having **a** sequence as set forth in nucleotides 1 to 20 of SEQ ID NO:'.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 1-5 and 13 are rejected under 35 U.S.C. 103(a) as being unpatentable over Rutledge et al (1991, as cited in the IDS) in view of Hattori et al (1995, as cited in the IDS), and Sathasivan et al (1991).

Rutledge et al teaches the nucleic acid and deduced amino acid sequence of the *Brassica napus* AHAS1 and AHAS3 genes (Fig. 2A and 2C). The reference teaches that DNA was isolated from leaf nuclei, relevant to claim 1 step (a). The reference also teaches that imidazolinone herbicides act through inhibition of AHAS (p.39, left column, last paragraph), and further teaches that herbicide resistance in *B. napus* mutants results from two unlinked alleles, and that the effect of combining the alleles in a hybrid line is additive for imidazolinone resistance. The reference teaches that the imidazolinone resistance alleles correspond to AHAS1 and AHAS3 (p.39, left column, last paragraph), and concludes that the sequences of the AHAS genes provides the basic information essential for the analysis of *Brassica* mutants with resistance to herbicides that act on AHAS (p.39, right column, last paragraph).

Rutledge et al does not indicate the nature of the mutations in AHAS 1 (PM1) and AHAS3 (PM2) that confer resistance to imidazolinone.

Hattori et al 1995 teaches the analysis of the AHAS3 gene from imidazolinone-resistant mutant *B. napus* cells, relevant to claim 1 step (c) of the instant application. The reference teaches that the AHAS3 gene from the mutant cells was cloned and sequenced, and the sequence of the gene from the mutant was compared to the wild-type AHAS3 sequence (p.420, right column, l.28). Hattori teaches the identification of a single basepair change (G to T) in AHAS3 that predicts a tryptophan to leucine amino acid change (p.421, left column, last paragraph), and provides a comparative alignment of deduced amino acid sequences in the region of the AHAS3 mutation responsible for herbicide resistance (p.421 Fig. 2). Based on the alignment provided in Fig. 2, and the

sequence of the AHAS3 gene provided by Rutledge et al, it is evident that the G to T mutation taught by Hattori is equivalent to the PM2 mutation claimed in the instant application. Hattori concludes that the identified mutation site in the AHAS3 gene is involved in the binding of imidazolinone herbicides, and teaches that the recovery of the same mutation in tobacco and *B. napus*. Further relevant to claim 4 of the instant application, Hattori teaches the amplification of the AHAS1 gene from isolated genomic DNA prior to determining whether or not mutations are present.

Relevant to claim 13, Hattori et al (1995) teaches a method in which primers are used to amplify the AHAS gene (p.420, right col.), where such primers satisfy the broadly claimed limitations of 'an oligonucleotide having a sequence as set forth in nucleotides 1 to 20 of SEQ ID NO: 66', as required by the claim.

Sathasivan et al teaches the analysis of an *A. thaliana* mutation in the acetolactate synthase gene (referred to within the reference as ALS, which is an art recognized synonym for AHAS). The reference teaches that the mutation provides the molecular basis for imidazolinone resistance in *A. thaliana* (p.1044 – Abstract). Sathasivan et al teaches the specific nature of the *A. thaliana* mutation responsible for herbicide resistance as a G to A single-point mutation at nucleotide 1958 of the coding sequence, which predicts a serine to asparagine substitution at amino acid 653 (p.1044, left column, last paragraph; Fig. 2; Table 1). Based on the nucleic acid sequence provided by Sathasivan et al (Fig 2B), the sequence of the AHAS1 gene provided by Rutledge et al as well as the teachings of Rutledge et al that the imidazolinone resistance alleles correspond to AHAS1 and AHAS3, and the fact that Hattori et al

teaches a mutation in AHAS3, it is evident that *A. thaliana* G to A mutation taught by Sathasivan is equivalent to the PM1 mutation claimed in the instant application. The reference also teaches that similar mutations at corresponding nucleotide positions of other acetolactate synthase genes can confer imidazolinone resistance (p.1049, left column, last paragraph). Further relevant to claim 5, Sathasivan et al teaches the analysis of the sequence of the acetolactate synthase gene using a chain termination method (p.1045 – Nucleic acid techniques; Fig. 2), which is a primer extension based method for sequencing that can detect single nucleotide polymorphisms.

It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have combined the information and methods provided in the cited references to have created the claimed invention of a method to assay for the presence or absence of the PM1 and PM2 mutations to determine the imidazolinone tolerance of a plant. One would have been motivated to develop such an assay to efficiently determine the relative level of herbicide resistance in a plant using molecular techniques based on the teachings of Rutledge et al, which teaches that combining resistance alleles in a hybrid line has an additive effect on resistance to imidazolinone. One would have had a reasonable expectation of success because the cited references teach both the general aspects of the properties responsible for imidazolinone resistance, as well as the specific molecular characteristics that confer imidazolinone herbicide resistance. Rutledge et al teaches that the two alleles responsible for imidazolinone resistance in a *B. napus* mutant correspond to the AHAS1 and AHAS3 genes, and that the effect of combining the alleles in a hybrid line is additive for

imidazolinone resistance (p.39, right column, last paragraph). Rutledge et al further teaches the nucleic acid sequences and deduced amino acid sequences of the *B. napus* AHAS1 and AHAS3 genes (Fig. 2A and 2C). Hattori et al teaches the identification of a G to T (tryptophan to leucine) mutation in the *B. napus* AHAS3 gene responsible for imidazolinone resistance that is equivalent to the PM2 mutation of the instant application. Sathasivan et al teaches the identification of a G to A (serine to asparagine) mutation in the *A. thaliana* ALS gene and provides a nucleic acid sequence indicating that this mutation is equivalent to the PM1 mutation of the instant application. It would be obvious to look for a G to A mutation at the PM1 position of the AHAS1 gene based on the teachings of Rutledge et al that there are two mutations that confer imidazolinone resistance and that one mutation is in AHAS1 and the other is in AHAS3 and the teachings of Hattori et al 1995 which teach that there is an imidazolinone resistance-conferring mutation in AHAS3 (which is identical to PM2). Thus based on Rutledge et al in view of Hattori et al 1995, it would be obvious to look for another mutation in the AHAS1 gene, and based on the teachings of Sathasivan et al it would be obvious to look for the G to A (Ser to Asn) mutation that is PM2.

Thus, in view of the teachings of the prior art, the claimed invention is prima facie obvious.

Claims 1-5, 12 and 13 are rejected under 35 U.S.C. 103(a) as being unpatentable over Rutledge et al (1991, as cited in the IDS) in view of Hattori et al (1995, as cited in the IDS), and Hattori et al (1992).

Rutledge et al teaches the nucleic acid and deduced amino acid sequence of the *Brassica napus* AHAS1 and AHAS3 genes (Fig. 2A and 2C). The reference teaches that DNA was isolated from leaf nuclei, relevant to claim 1 step (a). The reference also teaches that imidazolinone herbicides act through inhibition of AHAS (p.39, left column, last paragraph), and further teaches that herbicide resistance in *B. napus* mutants results from two unlinked alleles, and that the effect of combining the alleles in a hybrid line is additive for imidazolinone resistance. The reference teaches that the imidazolinone resistance alleles correspond to AHAS1 and AHAS3 (p.39, left column, last paragraph), and concludes that the sequences of the AHAS genes provides the basic information essential for the analysis of *Brassica* mutants with resistance to herbicides that act on AHAS (p.39, right column, last paragraph). Further relevant to claim 5, Rutledge et al teaches the analysis of the sequences of the AHAS genes using di-deoxy sequencing with primers (p.32 – DNA sequence analysis), which is a primer extension based method for sequencing that can detect single nucleotide polymorphisms.

Rutledge et al does not indicate the nature of the mutations in AHAS 1 (PM1) and AHAS3 (PM2) that confer resistance to imidazolinone.

Hattori et al 1995 teaches the analysis of the AHAS3 gene from imidazolinone-resistant mutant *B. napus* cells, relevant to claim 1 step (c) of the instant application. The reference teaches that the AHAS3 gene from the mutant cells was cloned and sequenced, and the sequence of the gene from the mutant was compared to the wild-type AHAS3 sequence (p.420, right column, l.28). Hattori teaches the identification of a

single basepair change (G to T) in AHAS3 that predicts a tryptophan to leucine amino acid change (p.421, left column, last paragraph), and provides a comparative alignment of deduced amino acid sequences in the region of the AHAS3 mutation responsible for herbicide resistance (p.421 Fig. 2). Based on the alignment provided in Fig. 2, and the sequence of the AHAS3 gene provided by Rutledge et al, it is evident that the G to T mutation taught by Hattori is equivalent to the PM2 mutation claimed in the instant application. Hattori concludes that the identified mutation site in the AHAS3 gene is involved in the binding of imidazolinone herbicides, and teaches that the recovery of the same mutation in tobacco and *B. napus*. Further relevant to claim 4 of the instant application, Hattori teaches the amplification of the AHAS1 gene from isolated genomic DNA prior to determining whether or not mutations are present.

Relevant to claim 13, Hattori et al (1995) teaches a method in which primers are used to amplify the AHAS gene (p.420, right col.), where such primers satisfy the broadly claimed limitations of 'an oligonucleotide having a sequence as set forth in nucleotides 1 to 20 of SEQ ID NO: 66', as required by the claim.

Hattori et al 1992 teaches the analysis of an imidazolinone resistance-conferring mutation in the AHAS gene of *A. thaliana*. The reference teaches that the mutation responsible for imidazolinone resistance is a G to A transition that predicts a Ser to Asn substitution in the amino acid sequence Ile Pro Ser Gly Gly (p.169 – Nucleotide sequence of imr1), and further teaches that the Ser that is substituted shows perfect conservation in all of the known wild-type plant AHAS genes including *B. napus*. provides the molecular basis for imidazolinone resistance in *A. thaliana* (p.1044 –

Abstract). Based on the nucleic acid sequence (Fig 1C) and amino acid sequence context (Fig 1C; p.169 – Nucleotide sequence of imr1) provided by Hattori et al 1992, the sequence of the AHAS1 gene provided by Rutledge et al as well as the teachings of Rutledge et al that the imidazolinone resistance alleles correspond to AHAS1 and AHAS3, and the fact that Hattori et al 1995 teaches a mutation in AHAS3, it is evident that the *A. thaliana* G to A mutation taught by Hattori et al 1992 is equivalent to the PM1 mutation claimed in the instant application.

Relevant to claim 12, Hattori et al (1992) teaches a method in which primers are used to analyze the AHAS gene (Figure 1B), where such primers satisfy the broadly claimed limitations of 'an oligonucleotide having a sequence as set forth in nucleotides 1 to 20 of SEQ ID NO: 24', as required by the claim.

It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have combined the information and methods provided in the cited references to have created the claimed invention of a method to assay for the presence or absence of the PM1 and PM2 mutations to determine the imidazolinone tolerance of a plant. One would have been motivated to develop such an assay to efficiently determine the relative level of herbicide resistance in a plant using molecular techniques based on the teachings of Rutledge et al, which teaches that combining resistance alleles in a hybrid line has an additive effect on resistance to imidazolinone. One would have had a reasonable expectation of success because the cited references teach both the general aspects of the properties responsible for imidazolinone resistance, as well as the specific molecular characteristics that confer imidazolinone

herbicide resistance. Rutledge et al teaches that the two alleles responsible for imidazolinone resistance in a *B. napus* mutant correspond to the AHAS1 and AHAS3 genes, and that the effect of combining the alleles in a hybrid line is additive for imidazolinone resistance (p.39, right column, last paragraph). Rutledge et al further teaches the nucleic acid sequences and deduced amino acid sequences of the *B. napus* AHAS1 and AHAS3 genes (Fig. 2A and 2C). Hattori et al 1995 teaches the identification of a G to T (tryptophan to leucine) mutation in the *B. napus* AHAS3 gene responsible for imidazolinone resistance that is equivalent to the PM2 mutation of the instant application. Hattori et al 1992 teaches the identification of a G to A (Ser to Asn) mutation in the *A. thaliana* ALS gene and provides the amino acid sequence context of the alteration (i.e. Ile Pro Ser Gly Gly changed to Ile Pro Asn Gly Gly) indicating that this mutation is equivalent to the PM1 mutation of the instant application. It would be obvious to look for a G to A mutation at the PM1 position of the AHAS1 gene based on the teachings of Rutledge et al that there are two mutations that confer imidazolinone resistance and that one mutation is in AHAS1 and the other is in AHAS3 and the teachings of Hattori et al 1995 which teach that there is an imidazolinone resistance-conferring mutation in AHAS3 (which is identical to PM2). Thus based on Rutledge et al in view of Hattori et al 1995, it would be obvious to look for another mutation in the AHAS1 gene, and based on the teachings of Hattori et al 1992 it would be obvious to look for the G to A (Ser to Asn) mutation that is PM2.

Thus, in view of the teachings of the prior art, the claimed invention is prima facie obvious.

Response to Remarks

Applicant has traversed (Remarks of 2/27/2007) the rejection of claims 1-5 as unpatentable over Rutledge et al in view of Hattori et al and Sathasivan et al.

Applicant initially remarks that Rutledge et al refers to the greater than expected complexity of the AHAS gene family (page 4, last paragraph), and that Rutledge et al is deficient in teachings of the PM1 and PM2 mutations. While the Examiner agrees that Rutledge does teach such complexity (i.e. three AHAS genes), in the rejection at hand, it must be recognized that Rutledge does specifically point to AHAS1 and AHAS3 as genes in which imidazolinone resistance-related mutations occur, and that it is the secondary references of Hattori et al and Sathasivan et al with provide the specific molecular basis of the mutations identified as PM1 and PM2 of the instant application.

Applicant remarks (page 5) that Hattori et al (1995) discloses the PM2 mutation but is deficient in any teachings of the PM1 mutations of *B. napus*. However, the Examiner has cited Hattori et al (1995) for its clear teaching of the molecular nature of the PM2 mutation in AHAS3, as recognized by Applicant, not for any teaching of the PM1 mutation.

Applicant further remarks (page 5) that Hattori et al (1995) is devoid of any teaching of a relationship between the disclosed mutation ad 'commercially relevant levels of imidazolinone tolerance' in *Brassica*. It is thus relevant to point out a definition of 'commercially relevant imidazolinone tolerance' is provided in the instant specification at page 5 paragraph [027]. The specification defines the 'commercially relevant imidazolinone tolerance' as the level of tolerance to imidazolinone herbicides exhibited by CLEARFIELD® canolas which contains both the PM1 and PM2 mutations. Thus any method of determining the presence of the PM1 and PM2 mutations is necessarily determining 'commercially relevant imidazolinone tolerance' because this is an inherent property of the presence of the mutations, as defined by the specification.

Applicants further point to the teachings of Hattori et al (1995) concerning the multiple mutations in the different AHAS genes which may contribute to herbicide resistance as evidence of the difficulty in defining the molecular basis of the PM1

mutation. However, these teachings of Hattori are not relevant with regard to identification of the PM1 mutation. Hattori et al (1995) indicates that multiple sites in that AHAS protein bind to different herbicides (page 424 left col.) and that the small amount of AHAS protein in plants makes purification and analysis of the protein difficult. Such teachings do not discount the value of the teachings of Hattori et al (1995) with regard to the PM2 mutation, and do not teach away from the findings of Sathasivan et al with regard to another imidazolinone resistance mutation.

Applicant argues that Sathasivan et al fails to cure the deficiencies of Rutledge et al and Hattori et al (1995) as applied to the PM1 mutation. Applicants point to Table I of the reference and argue that Sathasivan et al actually disclose several mutations that one could believe to be analogous to the PM1 mutation in *Brassica*. This argument is not found persuasive because, while the reference discloses five mutations in Table I, an analysis of those disclosed mutations shows that the proline-codon disrupting mutations do not provide imidazolinone resistance (instead to sulfonylurea), and the tryptophan-codon disrupting mutation (TGG to TTG / Trp to Leu) appears analogous to the PM2 mutation taught by Hattori et al (1995). Furthermore, such disclosure of additional mutations does not take away from the value of the teachings of Sathasivan et al with regard to the TGG to TTG / Trp to Leu mutation providing imidazolinone resistance.

Applicants conclude that given the complexity of the *Brassica* gene family disclosed in Rutledge et al, the number of possible herbicide resistance mutations in AHAS and the difficulty in defining the AHAS enzyme structure, the identity of the PM1 mutation would not have been evident. This is not found to be persuasive because, as discussed above, though Rutledge et al comment on AHAS gene complexity in *Brassica*, the reference does in fact provide all the nucleic acid sequences of all three AHAS genes, as well as the direct teaching that imidazolinone resistance mutations are found in AHAS 1 and AHAS3. That Hattori et al (1995) teaches that multiple sites within the AHAS protein interact with herbicides does not take away from the specific teaching of Hattori et al (1995) that a single particular mutation (the PM2 mutation in AHAS3) is involved in imidazolinone resistance. Thus after considering Rutledge et al in view of

Hattori et al (1995), one is left to identify a mutation in AHAS1, where the teachings of Sathasivan et al, as discussed above, direct one of ordinary skill in the art to the mutation of PM1 as disclosed in the instant application. Thus Applicants argument (page 6 of Remarks) that none of the references provides direction as to which of the many possible AHAS mutations could be PM1 is not found to be persuasive; the references of the rejection clearly point to the analysis of two mutations in AHAS1 and AHAS3 of *Brassica*, where the mutations are the PM1 and PM2 mutations of the instant application.

Applicant has traversed (pages 6 and 7) the rejection of claims 1-5 as unpatentable over Rutledge et al in view of Hattori et al (1995) and Hattori et al (1992).

Response to the traversal with regards to the teachings of Rutledge et al in view of Hattori et al (1995) has been presented earlier in this Response to Remarks section of the Office Action.

Applicant argues that Hattori et al (1992) fails to specify the identity of the PM1 mutation. This is not found to be persuasive because such an argument fails to take into account the teachings of all of the references. Rutledge et al teaches the nucleic acid and deduced amino acid sequences of the AHAS1 and AHAS3 genes of *Brassica*, and further teaches that two mutations, one in each gene, result in resistance to imidazolinone, and the two mutations together are additive for imidazolinone resistance. Hattori et al (1995) clearly teaches the nature of one imidazolinone resistance mutation as a G to T mutation resulting in a Trp to Leu substitution in the AHAS3 gene that in the PM2 mutation of the instant application. Thus, after considering Rutledge et al in view of Hattori et al (1995) one is left to identify a mutation in the AHAS1 gene that causes imidazolinone resistance. Hattori et al (1992) teaches the nature of a mutation in the AHAS gene of *A. thaliana* that is a G to A mutations resulting in a Ser to Asn substitution in the conserved amino acid context Ile Pro Ser Gly Gly. Hattori et al (1992) further teaches the conservation of the substituted Ser residue in other plants including *Brassica*. It is thus obvious to analyze the G to A mutation resulting in a Ser to

Asn substitution in the conserved amino acid context Ile Pro Ser Gly Gly in the *Brassica* AHAS1 gene, which is the PM1 mutation if the instant application.

And while applicants argue that none of the references cited in the rejections of claims under 35 USC 103 provide direction as to which of the many possible AHAS mutations could be PM1, nor do they indicate which of many possible parameters would have been critical to determine the identity of PM1, the Examiner maintains that in light of the cited references, one of skill in the art would be motivated to analyze the required nucleotide content of the PM1 mutation based on the teachings provided in the references. The cited references do not in fact supply 'many possible AHAS mutations' that could be PM1, but instead are directed to the PM1 mutation of the instant claims. And because the definition of 'commercially relevant imidazolinone tolerance' provides that such an indication is inherent to the presence of the two mutations, the Examiner maintains that it would not merely be 'obvious to try' the claimed method comprising determining the presence of the PM1 and PM2 mutations, but identification of the required mutations would in fact accomplish the claimed method.

The rejections are **MAINTAINED.**

Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., In re Berg, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); In re Goodman, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); In re Longi, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); In re Van Ornum, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); In re Vogel, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and In re Thorington, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 1-5, 12, and 13 are provisionally rejected on the ground of nonstatutory double patenting over claims 2-10, 12-21, 24, and 25 of copending Application No. 10/695,546 (Pub. No.: US 2004/0171027 A1). This is a provisional double patenting rejection since the conflicting claims have not yet been patented.

The subject matter claimed in the instant application is fully disclosed in the referenced copending application and would be covered by any patent granted on that copending application since the referenced copending application and the instant application are claiming common subject matter, as follows:

The copending '546 application claims methods for assaying a *Brassica* plant for imidazolinone resistant comprising the steps of isolating genomic DNA from a plant and detecting the PM1 mutation in AHAS1 and the PM2 mutation in AHAS3. The claims of the copending application also encompass the amplification of the isolated DNA (relevant to claim 4 of the instant application), as well as methods to detect single nucleotide polymorphisms that utilize the extension of primers (relevant to claim 5 of the instant application). Although the copending '546 application cites different nucleotide positions (paragraphs [0028]-[0029]) of the G to A mutation in AHAS1 and the G to T mutation in AHAS3, which are PM1 and PM2 respectively, it is evident from a

comparative alignment of the gene sequences in the copending applications that the identical mutations at the equivalent positions are claimed.

Furthermore, there is no apparent reason why applicant would be prevented from presenting claims corresponding to those of the instant application in the other copending application. See *In re Schneller*, 397 F.2d 350, 158 USPQ 210 (CCPA 1968). See also MPEP § 804.

Response to Remarks

Applicant has expressed a willingness to provide a Terminal Disclaimer over Application 10/695,546 upon indication of allowability of the claims of the instant application (Remarks page 7). As allowability of the claims is not yet indicated, the rejection is **MAINTAINED**.

Conclusion

No claim is allowed.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Stephen Kapushoc whose telephone number is 571-272-3312. The examiner can normally be reached on Monday through Friday, from 8am until 5pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached at 571-272-0735. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Patent applicants with problems or questions regarding electronic images that can be viewed in the Patent Application Information Retrieval system (PAIR) can now contact the USPTO's Patent Electronic Business Center (Patent EBC) for assistance. Representatives are available to answer your questions daily from 6 am to midnight (EST). The toll free number is (866) 217-9197. When calling please have your application serial or patent number, the type of document you are having an image problem with, the number of pages and the specific nature of the problem. The Patent Electronic Business Center will notify applicants of the resolution of the problem within 5-7 business days.

Applicants can also check PAIR to confirm that the problem has been corrected. The USPTO's Patent Electronic Business Center is a complete service center supporting all patent business on the Internet. The USPTO's PAIR system provides Internet-based access to patent application status and history information. It also enables applicants to view the scanned images of their own application file folder(s) as well as general patent information available to the public.

For all other customer support, please call the USPTO Call Center (UCC) at 800-786-9199.

Stephen Kapushoc
Art Unit 1634

BJ FORMAN, PH.D.
PRIMARY EXAMINER